Data Sheet (Cat.No.TMAY-03081)

TargetMol

Anti-BMPR1A/ALK-3 Antibody (6X147)

Product Details

Ig Type:	Rabbit IgG
Reactivity:	Mouse
Conjugation:	Unconjugated
Clone:	6X147
Purification:	Protein A

Applications

Application:	ELISA
Recommended	ELISA: 1:5000-1:10000

Properties

Stability & Storage:	Store at 2°C-8°C for 1 month. Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles. Preservative-Free.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	Recombinant Protein: Mouse ALK-3 / BMPRIA protein (TMPY-01374)
Antigen Species:	Mouse
Synonyms:	bone morphogenetic protein receptor, type IA;ACVRLK3;ALK3;CD292;10q23del;SKR5

Research Background

Activin receptor-Like Kinase 3 (ALK-3), also known as Bone Morphogenetic Protein Receptor, type IA (BMPR1A), is a type I receptor for bone morphogenetic proteins (BMPs) which belong to the transforming growth factor beta (TGF-β) superfamily. The BMP receptors form a subfamily of transmembrane serine/threonine kinases including the type I receptors BMPR1A and BMPR1B and the type II receptor BMPR2. ALK-3/BMPR1A is expressed in the epithelium during branching morphogenesis. Deletion of BMPR1A in the epithelium with an Sftpc-cre transgene leads to dramatic defects in lung development. ALK-3 and ALK-6 share a high degree of homology, yet possess distinct signaling roles. The transforming growth factor (TGF)-beta type III receptor (TbetaRIII) enhanced both ALK-3 and ALK-6 signaling. TbetaRIII associated with ALK-3 primarily through their extracellular domains, whereas its interaction with ALK-6 required both the extracellular and cytoplasmic domains. ALK-3 plays an essential role in the formation of embryonic ventral abdominal wall, and abrogation of BMP signaling activity due to gene mutations in its signaling components could be one of the underlying causes of omphalocele at birth. The type IA BMP receptor, ALK-3 was specifically required at mid-gestation for normal development of the trabeculae, compact myocardium, interventricular septum, and endocardial cushion. Cardiac muscle lacking ALK-3 was specifically deficient in expressing TGFbeta2, an established paracrine mediator of cushion morphogenesis. Hence, ALK-3 is essential, beyond just the egg cylinder stage, for myocyte-dependent functions and signals in cardiac organogenesis.

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